Spatial Consequences of Plant Induced Resistance to Herbivory

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Abstract

Induced Resistance in plants in response to herbivory has been both theorized and experimentally verified to affect the spatial distribution of herbivores. However, there has never been a theoretical or experimental consensus on whether induced resistance causes increased herbivore aggregation or increased evenness, as both have been experimentally documented. In recent years, theories regarding the benefit of induced resistance to plants has shifted from classical ideas of optimizing resource allocation towards a more eclectic set of theories often considering spatial and temporal plant variability and the distribution of herbivores among the plants. Therefore, we created a comprehensive model for induced resistance that describes a large number of plant-herbivore systems with induced resistance. We analyze the specific contributions of different aspects of induced resistance in order to uncover the mechanisms driving both herbivore aggregation and evenness. Furthermore, we investigate the population level consequences of these different herbivore distributions. Our model shows that induced resistance alone can cause both even and aggregated distributions of herbivores. With these results we are able to simply explain the apparent conflict in the literature between the experimental results indicating both increased herbivore aggregation and evenness in the presence of induced resistance. Additionally, we are able to show that both informed herbivore movement and plant-plant communication help to spread out herbivore damage, benefiting the plant population by sharing the risk of herbivory throughout the population.

Introduction

Over the last 40 years, induced resistance in plants in response to herbivory has been shown to greatly affect the behavior and performance of herbivores (Green and Ryan 1972, Schaller 2008). Here, we define induced resistance as inducible plant responses to herbivory that alter the preference, performance, or reproductive success of the attacker (Karban and Baldwin 1997). The time-scale of induced resistance can occur over periods shorter than the generation of the herbivore (rapid induced resistance) or longer (delayed induced resistance) (Haukioja and Neuvonen 1987). Inducible responses can affect herbivores directly by reducing the attacker's performance or preference for the host plant, or indirectly, by attracting or supporting antagonistic predators and parasites (Kessler and Halitschke 2007). There is also evidence that in some systems the induced resistance compounds are volatiles that are released to the immediate area (Heil and Karban 2010, Karban 2011). These volatile cues allow neighboring plants to preemptively trigger defense responses against expected future predation (plant-plant communication) (Baldwin et al. 2006) as well as influencing neighboring herbivores to avoid plants secreting these resistance compounds (informed herbivore movement) (Gómez et al. 2008).

The traditionally hypothesized benefit of induced resistance for plants postulates that resistance draws from a limited pool of resources, so limiting the expression of resistance to only occur in the presence of herbivory optimizes the allocation of resources for growth and reproduction (Kessler and Baldwin 2002). However, despite the attention to this *optimal-defense theory*, other reasons for the benefit of induced resistance have been theorized both in addition and in place of optimal defense (Agrawal and Karban 1999). One example of an alternate theory is that the temporal and spatial variability in plant traits caused by induced resistance is beneficial by itself, as the variability may reduce herbivore performance and ability to adapt (Adler and Karban 1994, Agrawal and Karban 1999). Another theory relies on the idea that induced resistance causes increases herbivore dispersion (Agrawal

and Karban 1999, Edwards and Wratten 1983). This describes induced resistance as a risk-spreading strategy for the plants, more evenly spreading the herbivory and its associated risks, so that each plant can tolerate the amount of herbivory that it suffers.

Many different herbivore-inducible traits have been investigated, both in terms of their direct chemical and behavioral effect on herbivores, and the temporal herbivore population dynamics in response to the induced resistance. However, less is understood about the population level spatial dynamics of either the herbivores or the plant damage. Understanding these spatial dynamics is essential to uncovering the ecological function of induced resistance. If induced resistance is able to increase herbivore dispersion or decrease plant damage throughout the population, plants could benefit from the induced resistance, regardless of the resource allocation. The idea that induced resistance could cause unexpected spatial distributions of herbivores was first proposed by Edwards and Wratten (1983). They proposed that induced resistance caused herbivores to move away from damaged plants, resulting in an over-dispersed distribution of herbivores (i.e. a distribution even than random). An opposing hypothesis suggests that induced resistance causes herbivores to move together from damaged to undamaged plants (or similarly move together avoiding damaged plants), resulting in an under-dispersed distribution of herbivores (i.e. a distribution more aggregated than random) (Underwood et al. 2005).

Both of these seemingly conflicting theories have support from empirical studies. Several studies, including the original Edwards and Wratten paper, have examined grazing patterns on leaves and the aggregation of damage at the end of a season, an indirect way of measuring the location of herbivores. These studies showed that in the presence of induced resistance, leaf damage was more over-dispersed than expected, allegedly as a result of an even distribution of insects (Edwards and Wratten 1983, Silkstone 1987). However, another study showed that while the distribution of herbivores always becomes more even over time, some systems exhibiting induced resistance result in more aggregated distributions than expected and more aggregated than in systems without inducible defenses (Underwood et al. 2005). A

fourth study showed both even and aggregated herbivore distributions, but always recorded aggregated damage (Bergelson et al. 1986). Thus induced resistance is shown to cause both even and aggregated herbivore distributions.

Three previous theoretical studies have attempted to model the spatial dynamics of herbivores in response to induced resistance (Lewis 1994, Morris and Dwyer 1997, Underwood et al. 2005). One model suggests that induced resistance does affect the spatial distribution of herbivores, but focused on how non-random herbivore movement effects the temporal dynamics of the system (Morris and Dwyer 1997). The second study investigates the aggregation patterns of herbivores in response to resistance, but assumed that herbivores have the inherent tendency to aggregate (Morris and Dwyer 1997). This model suggests that aggregation patterns can arise in an otherwise homogeneous environment, but did not isolate the influence of induced resistance. Only the recent model by Underwood et al. attempts to analyze if and how induced resistance alone can cause unexpected distributions of herbivores (Underwood et al. 2005). Underwood et al. created a simple model of induced resistance that explained the levels of aggregation present in their experimental system, which consisted of Mexican bean beetle larvae and soybean plants. However, this study did not comprehensively investigate the different aspects or mechanisms of induced resistance present across different systems and how they might cause the different aggregation patterns seen previously.

The purpose of the present paper is to create a general model to predict the different aggregation patterns of herbivores in the presence of induced plant resistance. We introduce a new model that we hope will clarify the means by which induced resistance causes both over and under-dispersion. Doing this we attempt to answer:

1. How does induced resistance affect the aggregation patterns of herbivory? Can induced resistance alone cause the differing spatial distributions (over-dispersed, under-dispersed, and random) seen empirically in different populations?

2. Which specific aspects of induced resistance are responsible for the differing aggregation patterns?

3. What are the population level implications of the resulting spatial arrangements for the herbivore and plant populations? Does herbivore aggregation affect the total damage to the plant population at the end of the season?

We approach these questions by creating a spatially explicit, individual based model of a plant-herbivore system with induced resistance. The model is simulated using realistic parameter ranges to determine the underlying cause behind the different spatial distributions of herbivores. As every parameter represents a specific aspect or mechanism of induced resistance, trends for each parameter provide insight into the underlying causes of the differing aggregation patterns. The population-level implications of these patterns are determined by examining the state of the plant population (i.e. total population damage and the distribution of damage) at the end of the season. While the model is designed to describe any system exhibiting induced resistance, it is fitted to field data from the system of Leaf Beetle larvae (*Trirhabda virgata*) in patches of Goldenrod (*Solidago altissima*). This will provide a biological foundation for viable parameter ranges as well as shed light on the dynamics of the specific system.

By doing a sensitivity analysis, we will show that induced resistance is able to explain both herbivore aggregation and evenness. These spatial distributions are dependent of the characteristics of the system, such as the herbivore population size, the time lag between damage and resistance induction, and the ability of herbivores to detect resistance. Specifically, herbivore population size has a large effect on the herbivore spatial distribution and alone can explain the different distributions that appear in the literature (Bergelson et al. 1986, Edwards and Wratten 1983, Silkstone 1987, Underwood et al. 2005). Additionally, we will show that both plant-plant communication and informed herbivore movement significantly affect the herbivore spatial distribution and the distribution of plant damage.

State Variable	Definition
$D(i,j)_t$	the damage of plant (i,j) at time t
$R(i,j)_t$	the resistance of plant (i,j) at time t
$H(i,j)_t$	the number of herbivores on plant (i,j) at time t

Table 1: Definitions of the functions used in the model

Methods

Model

The model is an agent-base, discrete-time model in a spatial framework. The spatial framework is a two-dimensional lattice. We only consider a single season, thus only rapid induced resistance is considered, limiting the analysis to changes occurring within a herbivore's lifetime. Therefore, plant and herbivore death and reproduction are ignored. The model is made up of three state variables, herbivores (H), plant damage (D), and plant resistance (R) (Table 1).

The model consists of two sets of rules. The first controls the resistance in the plants based on damage caused by herbivore feeding. The second controls the movement of the herbivores in response to the resistance. There are four different versions of the model:

1. simple induced resistance based on plant damage and random herbivore movement
2. plant-plant communication for resistance with random herbivore movement
3. no plant-plant communication but with informed herbivore movement
4. both plant-plant communication and informed movement

Damage and Resistance Rules

The damage of each individual plant on day t (D_t) is a function of damage already present (D_{t-1}) and the number of herbivores on the plant during the last time-step (H_{t-1}). The damage variable can be viewed as a percent of the plant damaged, and therefore ranges from 0 to 1. The plant's level of induced resistance to herbivory is modeled as being equal to the

damage on the plant at a given time, as long as the damage is above some damage threshold D_o. This allows for damage and resistance to be cumulative based on past damage and for resistance to decay over time, independent of current number of residing herbivores (Hare and Sun 2011). The model for damage and resistance dynamics is:

$$D(i,j)_t = D(i,j)_{t-1} + \alpha H(i,j)_{t-l} - \beta D(i,j)_{t-1}\Big(1 - D(i,j)_{t-1}\Big) \tag{1}$$

$$R(i,j) = \begin{cases} D(i,j)_t & \text{if } D(i,j)_t \geq D_o \\ 0 & \text{if } D(i,j)_t < D_o \end{cases} \tag{2}$$

where i and j are respectively the row and column indices of each plant.

Here, α is the rate the herbivores eat and β is the rate the plant repairs the damage, which is equivalent to the rate of decay of the induced resistance. The parabolic structure of damage decay as modeled by the the last term in equation (1): $-\beta D(i,j)_{t-1}\,(1 - D(i,j)_{t-1})$, simulates the assumption that plants do not divert resources to repair light damage and do not have the resources for repair when heavily damaged. This also ensures that fully damaged plants will not regrow spontaneously and helps to correctly simulate the exponential decay of resistance compounds (Hare and Sun 2011).

Damage and Resistance Rules Considering Plant-Plant Communication

When plant-plant communication is considered, if a plant is induced because of damage, the resistance of every uninduced neighbor is set to D_o. This is based on laboratory studies that show that the direct neighbors of an induced plant are also induced, but that undamaged plants induced by a neighbor do not further cause induced resistance in their neighbors

	Definition	Range
H	the number of larvae in the patch	(50,950)
D_o	the damage threshold for induced resistance	(0,0.5)
α	the harvesting rate of herbivores, *per herbivore per day*	(0.001,0.019)
β	the decay rate of damage, or the growth rate of the plants, *per day*	(0,1)
τ	The time lag between plant damage and an induced response, *days*	(0,35)
p_u	the probability a herbivore moves off of an uniduced plant	(0,0.24)
p_i	the probability a herbivore moves off of an induced plant	(0.3,1)
c	the probability a herbivore chooses correctly	(0.3,1)

Table 2: Definitions of the parameters used in the model

(Kessler 2013). In this case, the resistance equation changes to:

$$R(i,j) = \begin{cases} D(i,j)_t & \text{if } D(i,j)_t \geq D_o \\ D_o & \text{if } D(i,j)_t < D_o \text{ and } D(i,l)_t \text{ or } D(k,j)_t \geq D_o \\ 0 & \text{if } D(i,j)_t \text{ , } D(i,l)_t \text{ , } D(k,j)_t < D_o \end{cases} \tag{3}$$

where $k = i \pm 1$ and $l = j \pm 1$.

Movement Rules

We consider both random and directed movement in our model. For random herbivore movement, each herbivore in the model only considers whether or not there is induced resistance present in the host plant. Herbivores move at a higher rate away from an induced plant than from an uninduced plant. When the host plant is uninduced, each herbivore on the plant has a specified probability, p_u, of moving to one of its neighbors. When the host plant is resisting, each herbivore has a probability $p_i > p_u$ of moving to a neighboring plant chosen at random. The herbivores move individually and simultaneously.

The informed movement model retains the same probabilities of movement. However when the herbivores move, they have a probability c of moving to the least resisting neighbor and a probability of $(1-c)$ of moving randomly to one of the other neighbors. If there are two neighbors with equally low resistance or all neighbors are resisting equally, each herbivore

chooses randomly between them. This is based on data that shows that the herbivores will choose to move to an undamaged plant over a damaged one (Kessler 2013). As in the random movement model, herbivores move individually and simultaneously.

Parameterization and Simulations

All simulations were run over each parameter range (see Table 2) for 40 time steps (in days, around the length of the season where *Trirhabda* larvae are actively eating and moving) and 10 replicate simulations for each parameter combination.

The data to parameterize the model comes from a series of unpublished laboratory and field experiments run by André Kessler and his lab on the system of *Solidago altissima* larvae and *Trirhabda virgata* (Kessler 2013). In all runs of the model, except in the analysis of herbivore population size, the initial herbivore distribution is based on field data from *Trirhabda* and *Solidago* at the start of the season ($H = 355$). The plants in the model are laid out in 14x14 array. For each plant in the model, the initial number of herbivores on the plant was drawn at random from the data on the number of herbivores per plant at the start of the season. When analyzing the affect of the herbivore population size, the initial distribution was modeled to have a similar distribution as the data but with a different population size (Table 1). The outer two rows of plants are discarded before the analysis to negate unwanted edge effects. Simulations were run to confirm that the plot size has no effect on the final spatial distribution of the herbivores, provided that ratio of herbivores to plants remains constant [*data not shown*].

The parameter D_o, the induced resistance damage threshold, was estimated as 0.1 from data that shows the *Trirhabda* larvae move off of their host plant consistently when the plant reaches approximately 10% of leaf area damaged (Kessler 2013). The parameters α and β are not estimated from data. Therefore, both were analyzed over a broad range to ensure the inclusion of all realistic values. α was varied from 0.1% of a plant eaten per herbivore per day to 19%. β was tested over (0,1), its entire possible range, as $\beta = 0$ means resistance

never decays while $\beta = 1$ indicates instantaneous decay (resistance lasts for a maximum of 1 time-step). The parameter τ, the time lag between damage and induction, was shown to be around a day in this specific system (Kessler 2013), but a longer time lag does exist in other systems (Underwood et al. 2005). Therefore, τ was also varied over an extreme range from instantaneous inductions ($\tau = 0$) to 35 days.

The probabilities a herbivore moves off its host were estimated from an experiment that placed larvae on induced and uninduced plants to record the time it takes for the larvae to leave their respective host. On induced plants, 45% of the larvae move off each day. On uninduced plants, 16% of the larvae move the first day and 45% move subsequent days as the plant becomes induced. The probability of choosing correctly for the informed movement models was determined from an experiment showing that when given the choice between two plants, larvae chose an undamaged plant over a damaged one approximately 80% of the time.

Sensitivity Analysis

To analyze the contribution of each parameter on the resulting spatial distribution of herbivores, the model was run with each parameter varying over a specified range. During these analyses, all other parameters were fixed at the same value each time ($H \approx 355, \alpha = 0.01\beta = 0.11, D_o = 0.1, p_u = 0.16, p_i = 0.45, c = 0.8, \tau = 1$). These values were mostly derived from the *Trirhabda* and *Solidago* experiments, while α and β were chosen as reasonable estimations for the same system. For the parameters derived from experimentation, the range for each parameter was centered around the empirically derived value and extended in either direction to include biological extremes. τ, α, and β were estimated as broad ranges large enough to include all realistic values.

To separately test the influences of plant-plant communication and informed herbivore movement, only the informed movement model and the model with both informed movement and plant-plant communication were run. This is because varying c adjusts the influence of

informed movement on the model. When $c = 0.3$, its minimum value in our parameterization, herbivore movement is very close to random. This allows for a direct comparison between the models.

Aggregation Coefficient

To analyze the results, the spatial distribution of the herbivore populations is summarized by the aggregation coefficient $J = \sigma^2/\mu^2 - 1/\mu$, where μ and σ^2 are the mean and variance respectively of the number of herbivores per plant (Ives 1991). When $J = 0$, the herbivores are randomly distributed ($\sigma^2 = \mu$), when $J < 0$ they are more evenly distributed, and when $J > 0$ they are more aggregated than expected at random. The maximum value of J for a completely aggregated population, when every herbivore in the population resides on a single plant, is $N - 1 - 1/\mu$ (as $\sigma^2 = H^2/N - \mu^2 = (N-1)\mu^2$), where N is the number of plants in the patch ($N\mu = H$ =total number of herbivores in the patch). Maximal evenness occurs when $\sigma^2 = 0$ and gives the value of $J = -1/\mu$.

Based on sampling data from the beginning of the season, the herbivores always start the simulation slightly aggregated, around $J = 1.52$, as that is the distribution coefficient of the data used to generate the initial conditions. The maximum value of J for the average population size in these simulations is approximately equal to 98.72. As a completely even distribution is much close to a poisson distribution (random) for this population size, the minimum value of J is -0.282. For simulations run testing the correlation of population size and aggregation, the initial distribution of herbivores was generated to also have a distribution around $J = 1.52$.

The distribution of damage was simply characterized as the coefficient of variation, σ/μ. The aggregation coefficient J was not used, because the value of J depends on the units in which damage is measured (whereas for herbivores the unit is insects per plant, by assumption). This was chosen as it is a scale free measure of dispersion. For the coefficient of variation, an even distribution occurs when $\sigma/\mu = 0$ and a maximally aggregated distribution

occurs when $\sigma/\mu = \sqrt{N-1}$. However, we are not able to characterize a random distribution using the coefficient of variation as this occurs when $\sigma/\mu = 1/\sigma$, which varies in every simulation. Therefore, we are only able to analyze the distribution of damage comparatively (i.e. one simulation results in more or less aggregated damage than another).

Total Damage

In order to understand the effect of the different distributions on the health of the plant population, we also examine the total plant damage. Total damage is simply calculated as the sum of the damage to every plant at the end of the season. As the herbivore population size, herbivore harvesting rate, and damage decay rate are all constant throughout a single simulation, the only way total damage can change without changing one of those three parameters, is with differing distributions of the damage. Because of the parabolic structure of damage decay (Equation (1)), different loads of damage on a plant will cause the plant's damage to decay at different rates.

To reduce the influence of stochasticity, rather than analyzing the aggregation coefficients and total damage at the last time step of each simulation, equilibrium values were defined as the average value over the last five time-steps.

Results

Herbivore Population Size

This model was able to reproduce even, random, and aggregated distributions of herbivores with different realistic parameter combinations. However, during the sensitivity simulations for the other parameters the population size was fixed ($H \approx 355$) such that significantly even distributions are impossible simply because maximal evenness is too similar to random (Fig. 1(a)). For all simulations, the herbivore population size is constant over the course of the season. Within season mortality and reproduction are not considered.

There is a parabolic-like relationship between herbivore population size (relative to the

patch size) and aggregation in the informed movement models (Fig. 1(a)). Small herbivore populations produce more even distributions while large herbivore populations always end up completely randomly distributed. The smallest population size tested ($H = 50$), resulted in a even distribution of herbivores. Intermediate population sizes, similar to the population size used for the rest of the simulations, were able to produce more aggregation, depending on the model.

Plant-Plant Communication and Informed Movement

As c increases and movement becomes more informed, the herbivore distribution goes from random to aggregated (Fig. 1(b)). This is true regardless of whether plant-plant communication is included or not. When plant-plant communication was included however, the final herbivore populations were always less aggregated in distribution than without it. Both of these trends can also be seen in the other simulations. The herbivore distributions with informed movement never appreciably deviate from a random distribution. In addition, the herbivore distributions in the informed movement model almost always are more aggregated when there is no plant-plant communication (Figs. 1(a)-1(h)).

Feeding Rate and Resistance Decay

The parameters α, the herbivore harvesting rate, and β, the rate of damage decay, are very closely related in this model. With some simplification α can be viewed as the rate resistance increases per herbivore per day while β can be viewed as the rate of decay of the resistance per day. This is because damage and resistance are equal for $D > D_o$. Because of this relationship, α and β show fairly similar trends (Figs. 1(c),1(d)). When both are either very small or very large, the herbivore population ends up randomly distributed, but intermediate values in either ($0.003 < \alpha < 0.015, 0.1 < \beta < 0.4$) led to aggregation. Herbivore distributions were most aggregated when $0.004 < \alpha < 0.013$ and quickly dropped back to a random distribution for $\alpha > 0.015$. However, for larger values of β ($0.6 < \beta < 0.9$), the level of aggregation becomes even for all four models before returning to random as β

approaches one. The two models with plant-plant communication became significantly more evenly distributed than the two models without plant-plant communication.

Induction Time Lag and Damage Threshold

Herbivore aggregation increases with τ until $\tau \approx 15$ in the informed movement model and $\tau \approx 10$ for the model with both informed movement and communication(Fig. 1(h)). For values of τ greater than this, aggregation decreases, eventually reaching a random distribution at $\tau = 35$. The random movement models also show a slight increase in aggregation peaking around $\tau = 25$, though this is much less dramatic than with the informed movement models. Herbivore aggregation also increases as D_o increases, however only very slightly (Fig. 1(e)).

Movement Probabilities

The probability of a herbivore moving off of an uninduced plant p_u was the only parameter tested that showed no obvious correlation to herbivore aggregation (Fig. 1(f)). On the contrary, p_i, the probability of moving off of an induced plant, showed a strong positive correlation to herbivore aggregation in the informed movement models (Fig. 1(g)).

Total Damage

The only three parameters that show any correlation to total plant damage are the population size H, the decay rate of damage β, and the herbivore eating rate α. Both H and α show sigmoidal increases from no damage on the lower end of their ranges to 100% damage at the high end of their ranges (Figs. 2(a),2(c)). The trend for β is predictably opposite, with a exponential decrease from 100% damage at $\beta = 0$ to no damage when $\beta = 1$ (Fig. 2(d)). Every other parameter results in 55% to 60% average damage regardless of its value (Fig. 2).

Distribution of Damage

Opposite the trends in herbivore distribution, the random movement models almost always result in more aggregated damage than their informed movement counterparts (Fig. 3). This is true across the entire range every parameter except the time lag parameter τ (Fig. 3(h)) and

the high end of the range for p_i. Here the informed movement model is more aggregated than either of the two random movement ones as these decrease over the range of p_i (Fig. 3(g)).

In the two parameters that effect the rates of damage (H, α) the distribution of damage in the random movement models starts aggregated for small values of the respective parameter, and decreases towards a more even distribution as the parameter increases with a spike in aggregation around the same values as the herbivore distribution spiked in the informed movement models (Fig. 3(a),3(c)). The informed movement models follow the same trends in these parameters, although much less exaggerated. β, the only parameter that effects the rate of damage recovery for plants, follows an opposite trend, increasing in aggregation as β increases with an aggregation spike also around the same values as the herbivore distribution spiked in the informed movement models (Fig. 3(d)). The damage distribution decreases as c increases before stabilizing and even starting to increase slightly after $c = 0.6$ (Fig. 3(b)). For D_o and p_u, the random movement models retain high levels of aggregation over the whole range of the parameters; the informed movement model stays slightly aggregated, while the model with both informed movement and communication stays slightly evenly distributed (Fig. 3(e),3(f)).

Discussion

It has been long accepted that induced resistance can cause unexpected distributions of herbivores, yet the mechanisms behind these distributions is still largely a mystery. Here, we were successful in creating a generalized model of induced plant resistance to describe a wide range of systems and that reproduces even, random, and aggregated herbivore distributions. The model performed fairly well in comparison to the *Trirhabda* and *Solidago* data, which predicts an end of season aggregation coefficient $J = 0.27$ (Kessler 2013), well within the range of model's output and fairly close to the values returned from our baseline model parameterization ($J \approx 0.5$).

The ability of induced resistance to cause aggregation or evenness is highly dependent

on characteristics of the system, supported by the range of results discussed previously (Bergelson et al. 1986, Edwards and Wratten 1983, Silkstone 1987, Underwood et al. 2005). Underwood et al. found, that a time lag produces herbivore aggregation (Underwood et al. 2005). This is partially reinforced by the findings of our informed movement models as time lags short enough to allow for the system to respond before the end of the season caused significant herbivore aggregation (Fig. 1(h)). However, unlike the Underwood model, the aggregation in the random movement models only increased minimally and at very large time lag (τ) values.

Herbivore aggregation is not only dependent on a time lag. The population density of the herbivores has a very large effect on their distribution and alone is able to explain the discrepancies between the different studies (Fig. 1(a)). The three studies that reported over-dispersion of herbivory in response to induced resistance all investigated systems of insects or larvae on trees (Bergelson et al. 1986, Edwards and Wratten 1983, Silkstone 1987). In these cases, the plants in our model correspond to individual leaves or branches in the experiments as singular branches can respond to herbivory autonomously (Haukioja 1990). The population of leaves would greatly outnumber the herbivore population. The model with this low herbivore density would then correctly predict an even distribution of herbivores. Comparatively, the Underwood system of Mexican bean beetle larvae and soybean plants has a much higher herbivore population density, and again matched the models predictions with higher herbivore aggregation. These results are especially interesting in light of herbivore outbreaks, as it has been theorized that dispersion of herbivory could influence the timing of outbreaks or even the initial development of outbreak threshold levels (Kessler et al. 2012).

Traditional views of plant-plant communication are evolutionary troubling as the emission of these volatiles would assist a genetically distinct neighbor competing for the same resources, with no direct fitness advantage to the emitter (Baldwin et al. 2002). This has prompted a recent rebranding of plant-plant communication towards the *receiver* rather than the *emitter* of the signals, allowing for a much more palatable discussion of the evolution

of this phenomenon (Baldwin et al. 2002, Heil and Karban 2010). Nonetheless, simulations which vary the parameter c, the parameter that controls the effect of informed herbivore movement on the model, reveals that increased herbivore information always results in higher herbivore aggregation (Fig. 1(b)). Moreover, adding in plant-plant communication subsequently reduces that aggregation closer to random. These results can also be seen in the other model runs, as the informed movement model always results in the highest herbivore aggregation, followed by the model with both informed movement and plant-plant communication, while the random movement models both almost always result in a random distribution of herbivores (Fig. 1). This is due to the fact that informed herbivores will tend to move together from resisting plants to their undamaged neighbors. However, when plant-plant communication is considered the communicating plants will 'confuse' herbivore movement as all neighbors of a damaged plant will be induced as well, spreading out the herbivores between the neighbors of the damaged plant. Additionally, these neighbors will also always be resisting already, causing further herbivore movement and spread. When considering the fact that aggregated damage can be more detrimental to a plant population than that same damage dispersed throughout the population (Agrawal and Karban 1999), these results defend the theory of induced resistance as a risk-spreading strategy as plant communication helps disperse the herbivory more evenly among the plant population.

This is further supported when looking at the distribution of the damage, as the aggregation of damage decreases as the influence of informed herbivore movement increases (Fig. 3(b)). Similarly, when looking at the other parameters, the inclusion of plant-plant communication in the model always results in a more even distribution of damage, especially when informed movement is considered as well (Fig. 3). Interesting, unlike with the herbivore distribution, the random movement models almost always result in more aggregated damage than their informed movement counterparts (Fig. 3). This is because uninformed herbivores have no information about the plants they move to. This tends to result in the uninformed herbivores moving back and forth between highly damaged plants, creating 'pockets' of high

damage while other areas of the patch remain untouched [*data not shown*]. Conversely, informed herbivores often end up more aggregated but with much more even distribution of damage. This is probably because while the informed herbivores tend to move together, the groups damage each plant until its damage is on par with the surrounding plants, and then move on. Thus, by causing increased herbivore aggregation the presence of herbivore information would seem to counteract plant attempts to spread the risk of herbivory. However, more even damage distributions indicate herbivore information actually favors an individual plant goals of evenly distributing the risks of herbivory amongst its neighbors.

Despite the varying distribution of damage, the total plant damage is similar for all parameters except those directly changing the rates of damage (H, α) and damage recovery (β) (Fig. 2). This is because the total number of herbivores only fluctuates slightly (due to discarding the outer rows) and the herbivore feeding rate and plant recovery rate remain constant over the course of each simulation. The only variation in the total damage done is because the resistance decay is modeled by a non-linear function. With the constant population (no births, deaths, or migration) and constant herbivore harvesting rate assumptions of this model, induced resistance has little effect on the plant populations total damage at the end of a season. Therefore, the only benefit of induced over constitutive resistance must be contained in its ability to manipulate the herbivore and damage distributions. Future work should test this hypothesis with a more explicit evolutionary model. A game-theory approach to induced resistance may fully uncover whether induced resistance is a stable evolutionary strategy even if it is unable to significantly deter herbivore damage on a population level.

These results demonstrate the importance of creating a general model of induced resistance. With so many aspects of induced resistance able to alter the dynamics of the system, it is all the more important to view each system in a comprehensive way. The inclusive nature of our model will allow for future parameterizations of any of the large number of plant-herbivore systems with induced resistance. Our model shows that both aggregated

and even herbivore distributions can be explained by induced resistance. These seemingly conflicting experimental results in the literature are all a part of a bigger picture. Moreover, while induced resistance does cause higher levels of herbivore aggregation with the specific herbivore population density investigated, both plant-plant communication and informed herbivore movement help to spread the risks of herbivory, possibly explaining their ecological benefit for the plants.

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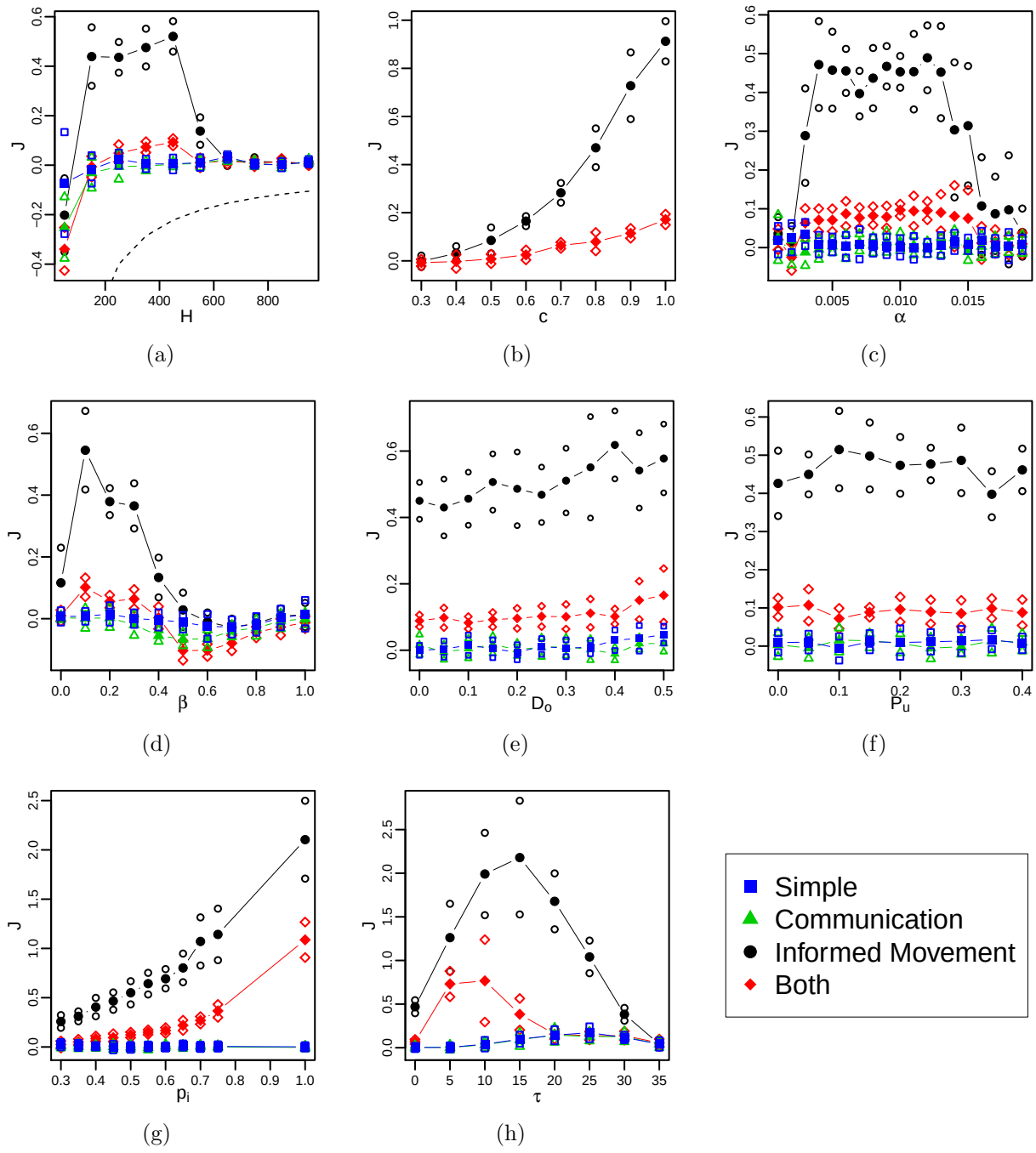

Figure 1: The equilibrium distribution values for varying values of each parameter. The solid points are the mean J value over 10 runs and the hollow points are one standard deviation from the mean. The dashed line in (a), the population size plot, represents the minimum possible value of J. The minimum and maximum values are fixed for every other plot at $J \approx -0.282$ and $J \approx 98.72$ respectively.
$\alpha = 0.01, \beta = 0.11, D_o = 0.1, p_u = 0.16, p_i = 0.45, \tau = 1, c = 0.8, H \approx 355$

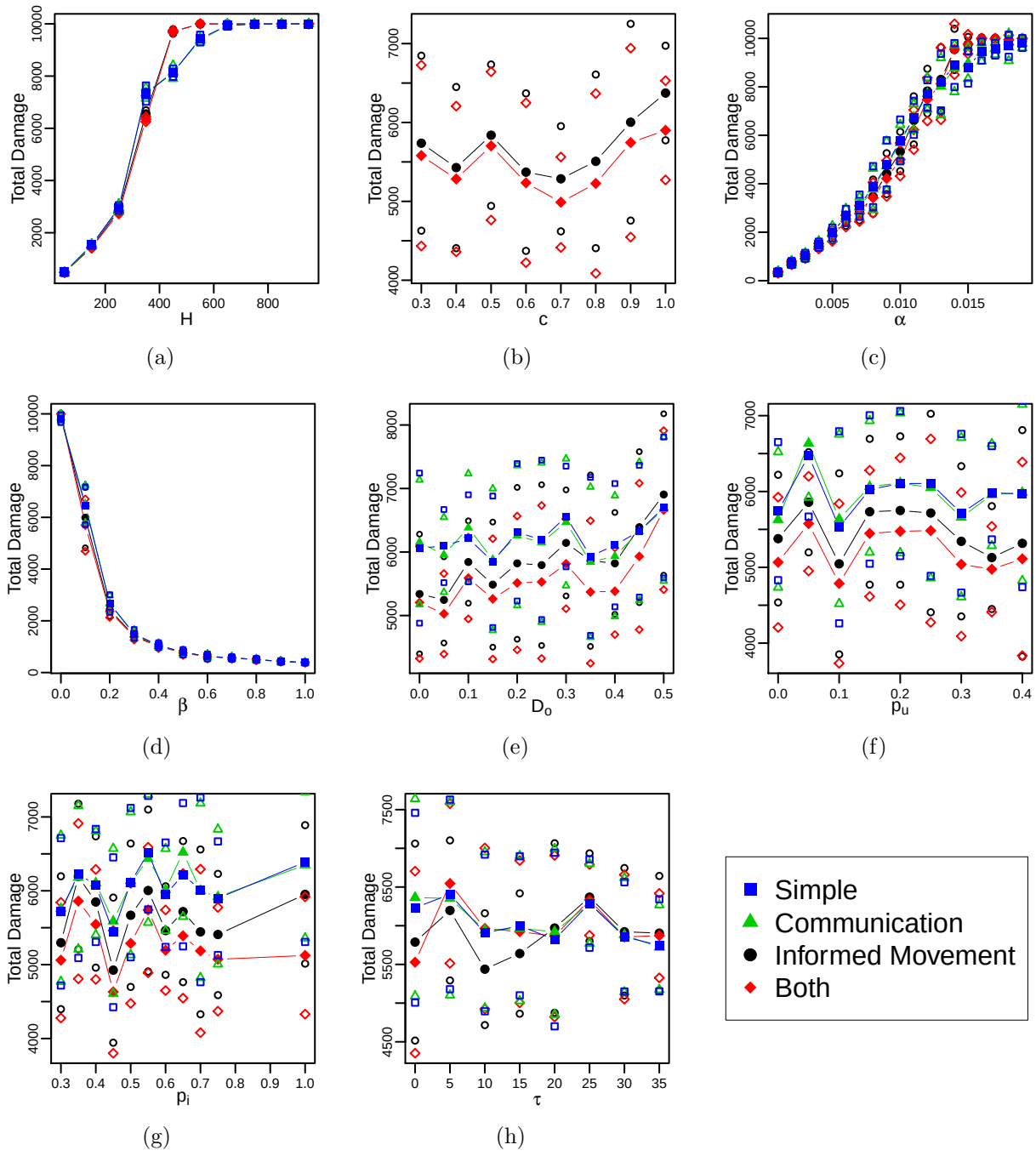

Figure 2: The total damage in the plant population for varying values of each parameter. The solid points are the mean damage over 10 runs and the hollow points are one standard deviation from the mean.
$\alpha = 0.01, \beta = 0.11, D_o = 0.1, p_u = 0.16, p_i = 0.45, \tau = 1, c = 0.8, H \approx 355$

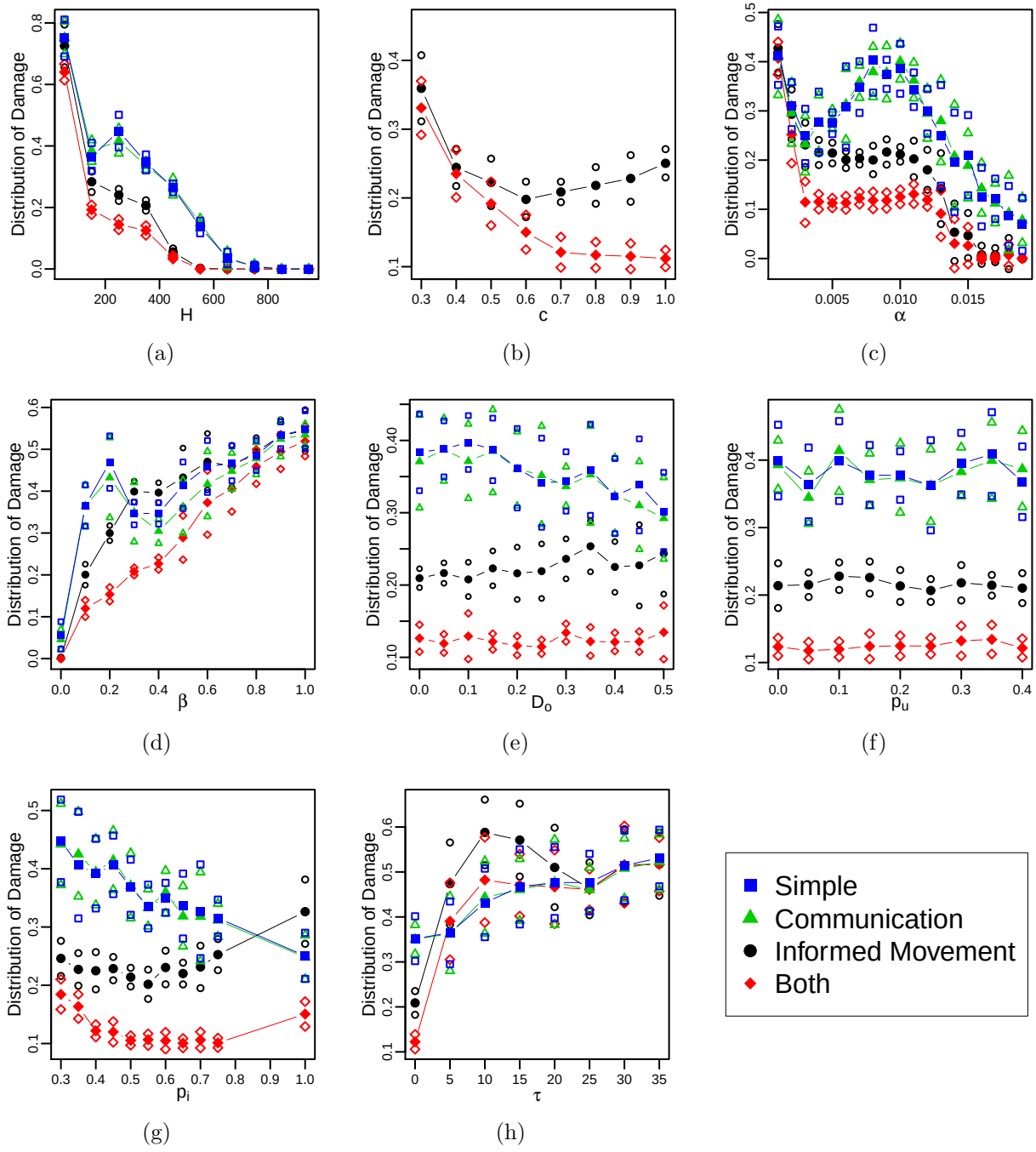

Figure 3: The equilibrium damage distribution values for varying values of each parameter. The solid points are the mean coefficient of variation value over 10 runs and the hollow points are one standard deviation from the mean.
$\alpha = 0.01, \beta = 0.11, D_o = 0.1, p_u = 0.16, p_i = 0.45, \tau = 1, c = 0.8, H \approx 355$